HISTOPATHOLOGICAL EVIDENCE FOR PULMONARY EMBOLI IN EXPERIMENTAL DECOMPRESSION SICKNESS DIAGNOSED BY RADIOISOTOPIC LUNG SCANNING

A.T.K. Cockett, M.D., S.M. Pauley, M.D., J.C. Saunders, M.D.

and A.P. Roberts, ~~M.D.~~ B.A.

From the Departments of Surgery/Urology, Harbor General Hospital, Torrance, California, 90509 and UCLA School of Medicine, Los Angeles and the Division of Urology, University of Rochester School of Medicine, Rochester, New York.

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ABSTRACT

Animals underwent experimental overcompression and decompression. Radioisotopic pulmonary scans were performed to diagnose aeroemboli. Biopsy of the cold areas were performed 48 hours after the chamber procedure and following dextran treatment. Pulmonary edema and hemorrhage are demonstrated.

During the past few years we have emphasized the importance of the lungs as a major target organ for gaseous embolization during moderately severe decompression procedures.[1-3] Experimental decompression sickness was induced in adult mongrel dogs by exceeding standard U.S. Navy decompression tables.

Mongrel dogs previously splenectomized were overcompressed to a barometric pressure of 73.5 PSIG. This pressure was maintained for sixty minutes. The animals were subsequently decompressed at the rate of 7 PSIG until surface was reached. No decompression stops were introduced. This experimental model is lethal for mongrel dogs.

Radioisotopic lung scanning methods were employed more recently to localize areas of pulmonary embolization. This procedure was accomplished by using macroaggregates of RISA ^{131}I. The aggregates had a mean particle size of 40 μ. We were impressed with the sensitivity and accuracy of the technique to detect significant pulmonary embolization. Adequate controls had to be instituted to demarcate and rule out the presence of artifacts.

The purpose of this report is to present histopathological evidence for the presence of pulmonary embolization. Presence of emboli was detected by serial lung scanning.

MATERIALS AND METHODS

We have previously outlined our techniques[4] for the production of aeroemboli. A decompression chamber (Figure 1), maintained by the United States Navy, was used in all of our experiments.

Two days prior to a scheduled overcompression-decompression procedure three or four animals were selected for pre-chamber pulmonary scanning. A Picker-Magna Scanner adapted for our animal procedures was employed. Three hundred microcuries of RISA macroaggregates were injected intravenously. Three views (left and right laterals and posterior positions) were obtained.

On the day of exposure to the chamber the same group of animals were anesthetized with intravenous pentobarbital. A large caliber endotracheal tube was inserted in each animal to allow free and voluntary respiratory exchange.

Immediately following the chamber run the animals were transported to the radioisotopic facility for a repeat lung scan.

When evidence for pulmonary emboli was observed on the lung scan immediately following the chamber run, a third lung scan was scheduled 48 hours later. A thoracotomy overlying the suspected areas was performed at this time, and multiple lung biopsies were made.

Conventional x-rays of the chest were obtained prior to and following chamber procedures.

RESULTS

Illustrative lung scans from four animals with the accompanying lung sections obtained by biopsy are included. The study covers a 3-year period.

Figure 2A is a composite of four sequential lung scans. Upper left is a pre-chamber control scan performed 2 days prior to decompression. Upper right is the post-chamber scan obtained 2 hours after decompression. Decreased radioactivity in the left apical region is seen. Lower left scan was obtained 48 hours after chamber removal. A reduction in radioactivity is still present. The lower right scan was obtained 4 weeks later. Normal activity is present. Figure 2B is a photomicrograph (100X) of a biopsy from the left apical region. Edema and hemorrhage are seen.

Figure 3A is a composite of four sequential scans. Upper left is a pre-chamber control scan. Upper right is a scan performed 3 hours after decompression. Decreased radioactivity in the left apex is seen. Lower left was obtained 48 hours after chamber removal. The lower right scan was performed 3 weeks later. Figure 3B is a photomicrograph (100X) of the biopsy made 48 hours after chamber removal. Edema and hemorrhage are evident.

Figure 4A is a composite of 3 scans obtained in a third animal. Scan on the left was obtained 2 days prior to the chamber run. The

middle scan was obtained 2 1/2 hours after the chamber run. Decreased radioactivity in the left apical region is seen. The scan on the right was obtained 48 hours later. The scan appears normal. Figure 4B is a photomicrograph obtained of the left apical area biopsied 48 hours after chamber removal. Hemorrhage and pulmonary edema are present.

Figure 5A is a composite of 2 scans. The scan on the right was obtained 3 hours after decompression. Decreased radioactivity on the left in the apex is seen. Figure 5B is a photomicrograph of this area obtained by biopsy 36 hours later. Pulmonary edema and hemorrhage are seen.

DISCUSSION

Since the radioisotopic lung scans can be misleading if adequate controls are not instituted, we felt compelled to biopsy the cold areas demarcated by lung scans. The lung sections clearly demonstrate areas of embolization. Hemorrhage and pulmonary edema comprise a major part of each field.

Conventional x-ray views of the lungs (PA and lateral views) were obtained before and after the chamber run to rule out the possibility of parenchymal disease. In dogs a pneumonitis can be a problem following experimental procedures.

The lung scan is still a screening test, and only large embolic areas are easily detected following the scanning procedures. We have employed color techniques[5] in an attempt to detect smaller embolic-cold areas. Again, we were impressed with the limitations of this technique. During the past two years we have used a modification of the lung scan. Radioactivity detected by the scintillation counter has been recorded on magnetic tape. We are evaluating computerized techniques in hopes of detecting smaller areas of embolization.

We are convinced that the venous systemic circulation gives rise and transports the aeroemboli to the lungs. We are also impressed with the presence of fatty emboli co-existing in the venous circulation. Both types of emboli bombard the pulmonary arterial circulation and are responsible for the "choking" symptoms which have been described. Number and size of both types of emboli dictate the course which the animal follows. If showers of emboli are widespread and severe, the animal succumbs. We have previously maintained an effective circulating blood volume by re-expanding the animal's plasma volume to normal levels.[6] Dextran (LMW and medium-70,000 molecular weight) has been effective in protecting animals from a lethal type of decompression sickness.[7] It would appear now that dextran is also beneficial because of other molecular actions. This colloid can effectively cope with the increased lipid content which apparently develops during fatty embolization. The effectiveness of dextran in lowering lipid levels has been previously described in rats.[8]

Still underway in our laboratory are selected studies to determine the types of lipids undergoing changes during decompression sickness. Heparin, because of its lipemic clearing effect, is also being evaluated as a therapeutic agent in treating experimental decompression sickness.

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